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Early linear growth velocity as a predictor of stunting at 24 months among infants in peri-urban Lusaka, Zambia: diagnostic and longitudinal analysis

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Research Article

Keywords: linear growth velocity, stunting, infants, longitudinal analysis, mixed-effects models, sub-Saharan Africa

Posted Date: June 9th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9703203/v1

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Additional Declarations: No competing interests reported.

Abstract

Background: Linear growth faltering remains a major contributor to childhood stunting, but the predictive value of early growth velocity is not well defined. This study assessed whether early linear growth velocity predicts stunting at 24 months and characterised growth-velocity trajectories among stunted and non-stunted children.

Methods: We analysed longitudinal anthropometric data from 120 children (enrolled at 6–8 weeks in a rotavirus vaccine trial in Lusaka, Zambia) with serial length/height and age measurements from enrolment through 24 months. Stunting at 24 months was defined as a height-for-age z-score <−2, using WHO growth standards. Linear growth velocity was calculated as the change in length/height in centimetres divided by the change in age in months across successive measurement intervals. The primary analysis assessed whether early growth velocity predicted 24-month stunting using receiver operating characteristic curves, area under the curve, and an optimal Youden-index cut-off, with sensitivity, specificity, predictive values, likelihood ratios, and accuracy estimated. Logistic regression examined unadjusted and adjusted associations. Longitudinal growth-velocity trajectories were compared by 24-month stunting status using mixed-effects models and spline-smoothed plots.

Results: Among 120 children, 118 had complete data for the primary prediction analysis; 58 (49.2%) were stunted at 24 months. Early growth velocity showed limited standalone discrimination for 24-month stunting (AUC 0.576). The optimal cut-off was 2.99 cm/month, with sensitivity 51.7%, specificity 63.3%, PPV 57.7%, NPV 57.6%, and accuracy 57.6%. In adjusted analysis, higher early growth velocity was associated with lower odds of stunting (aOR 0.63; 95% CI 0.42–0.93), and model discrimination improved substantially (AUC 0.810). Trajectory analyses showed generally lower growth velocity among children stunted at 24 months.

Conclusion: Early linear growth velocity alone showed limited screening utility for 24-month stunting, but longitudinal velocity patterns highlighted growth faltering periods that may inform targeted growth-monitoring interventions.

1. Introduction

Stunting, defined as a height-for-age z-score (HAZ) below -2 standard deviations from the World Health Organisation (WHO) child growth standard median, remains one of the most persistent forms of childhood undernutrition and reflects sustained linear growth failure during the most developmentally sensitive period of early life. In 2024, it was estimated that over 150 million children under the age of five were stunted worldwide, with the burden concentrated in sub-Saharan Africa (United Nations Children's Fund (UNICEF) et al., 2025), and is associated with impaired cognitive development, increased morbidity, reduced educational attainment, and lower adult economic productivity (Benjamin-Chung et al., 2023; de Onis and Branca, 2016; Mertens et al., 2023). In Zambia, almost a third of children below the age of five are stunted, and this number is much higher in rural areas (Zambia Statistics Agency, Ministry of Health (MOH) Zambia, and ICF, 2024). Although HAZ is widely used to classify stunting, it is a static indicator that identifies growth deficit after it has accumulated. Consequently, there remains an important evidence gap on whether early linear growth velocity, derived from repeated length/height measurements, can identify children at increased risk of being stunted by 24 months and thereby support earlier, more targeted nutrition and child health responses.

Linear growth velocity provides a dynamic measure of child growth because it captures the rate of length/height gain over defined age intervals, rather than relying only on attained height at a single time point. Current anthropometric indicators, such as HAZ, identify stunting only after substantial growth deficits have already occurred, limiting opportunities for early intervention and prevention (Leroy et al., 2014; Victora et al., 2010). Identifying children at risk before stunting becomes established is particularly important in low- and middle-income countries (LMICs), where the burden of undernutrition remains high and early-life growth faltering is associated with long-term adverse health

and developmental outcomes. Growth velocity, which reflects the rate of change in length over time, may provide a more sensitive and dynamic measure for detecting early growth faltering than static anthropometric indices (Cole, 2012; Leroy et al., 2014). Emerging evidence from LMICs suggests that poor early postnatal growth velocity is associated with later stunting and impaired cognitive development (MAL-ED Network Investigators, 2017; Victora et al., 2010). However, there remains limited evidence on the predictive utility of early linear growth velocity for subsequent stunting in Zambian infants. Establishing whether early growth velocity can identify children at increased risk of later stunting could support earlier targeting of nutritional and public health interventions during critical periods of child growth and development.

This study examined whether linear growth velocity during the first three months of life predicts stunting at 24 months among infants in peri-urban Lusaka, Zambia. It also characterised longitudinal growth-velocity patterns over the first two years of life and compared these trajectories between children who were stunted and those who were not stunted at 24 months.

2. Materials and Methods

Study design

This was a retrospective cohort study nested within an investigator-led, open-label, randomised controlled trial comparing two versus three doses of Rotarix^{TM} conducted in Kanyama township, a peri-urban community of Lusaka, Zambia. This setting is characterised by high population density, limited formal infrastructure, inadequate access to water and sanitation, and an increased burden of childhood infections, all of which are known to elevate the risk of early growth faltering. Infants in the original cohort were enrolled at 6-8 weeks and followed through 24 months of age, with anthropometric data collected during scheduled routine and sick visits.

The present analysis utilises only data from participants with complete anthropometric measurements at baseline, 3, and 24 months. We quantify linear growth velocity at multiple intervals during infancy and assess the predictive association between early linear growth and stunting at 24 months of age.

Study population

In this analysis, participants were included if they were recruited between 6-8 weeks, were healthy infants, as established by medical history and physical examination during enrolment in the original RCT, had a baseline anthropometric measurement, and a recorded length measurement at 24 months. Infants were excluded if they were lost to follow-up before 24 months of age.

Ethical considerations

Ethical approval for the parent study and subsequent secondary analysis was obtained from the University of Zambia Biomedical Research Ethics Committee (UNZABREC) (REF. No. 6630-2025) and the Zambia National Health Research Authority (NHRA) (2239/19/05/2025). Written informed consent was obtained from the parent or legal guardian of each participating infant before enrolment into the study. No personal identifiers were retained, and confidentiality guidelines were fully adhered to.

Variables and operational definitions

Stunting, the outcome of interest at 24 months, was defined as HAZ <−2 SD based on the WHO child growth standards. A binary variable was used for stunting. The primary exposure of interest was early linear growth velocity

from 0 to 3 months of age. Growth velocity was computed as the change in length (in cm) divided by the time interval (months) between measurements.

The covariates were selected based on the WHO stunting conceptual framework and on existing literature (World Health Organization, 2016). These included: infant characteristics: sex, HIV exposure, baseline stunting and malnutrition, and number of diarrhoeal adverse events (AEs) before 24 months. Maternal characteristics included maternal height (in meters), parity category, age, marital status, level of education, and employment status. Household characteristics included the type of sanitation facility (improved versus unimproved) and source of drinking water.

Statistical analysis

The sample size estimate was based on the Receiver Operating Characteristic (ROC)/area under the curve (AUC) objective. Under an assumption of a null AUC of 0.50 and assuming an anticipated AUC of 0.70, the minimum clinically relevant discrimination, α at a 0.05 significance level and 80% power, the minimum required sample size was 42 participants as cases and 42 as controls. Since the analysis was based on existing data from a clinical trial, all participants who met the eligibility criteria were included, providing substantially greater precision than the minimum required. Before analysis, records with missing height observations were dropped.

Baseline demographic and clinical characteristics were summarised using means (SD) or medians (IQR) for continuous variables, and frequencies (%) for categorical variables. The categories of stunting trajectory used in the analysis were based on the classification system presented by (Benjamin-Chung et al., 2023), which classified children by stunting trajectory. Stunting at each assessment point was defined as a HAZ below –2 standard deviations from the WHO Child Growth Standards median. The primary outcome was stunting status at 24 months. Linear growth velocity was calculated directly from measured length or height and age as the change in length or height in centimetres divided by the corresponding change in age in months. The primary exposure was early linear growth velocity from enrolment to approximately 3 months of age. Because infants were enrolled at 6–8 weeks, this measure represents early post-enrolment growth velocity rather than birth-to-3-month growth velocity. Additional interval-specific velocities were constructed for 3–6, 6–9, 9–12, 12–15, 15–18, 18–21, and 21–24 months to describe longitudinal growth patterns and examine whether later growth intervals were associated with stunting at 24 months.

Records with missing age or length/height measurements were excluded. Implausible anthropometric values were examined, and length/height measurements outside the biologically plausible range for infants and young children were excluded. For each child, baseline was defined as the earliest available non-missing length/height measurement. Scheduled follow-up measurements were constructed by selecting the measurement closest to each target age within prespecified age windows: 2–5 months for the 3-month visit, 5–8 months for the 6-month visit, 8–11 months for the 9-month visit, 11–14 months for the 12-month visit, 14–17 months for the 15-month visit, 17–20 months for the 18-month visit, 20–23 months for the 21-month visit, and 23–26 months for the 24-month visit. Children were included in the primary prediction analysis if they had complete data on stunting status at 24 months and early linear growth velocity from enrolment to approximately 3 months.

For the primary predictive analysis, ROC curve analysis was used to assess the ability of early linear growth velocity to discriminate between children who were stunted and those who were not stunted at 24 months. Because lower growth velocity was hypothesised to indicate a higher risk of subsequent stunting, the ROC analysis was based on the inverse of early growth velocity as the risk marker. Predictive performance was quantified using the area under the ROC curve with 95% confidence intervals. The optimal screening cut-off was identified using the Youden index,

defined as the sum of sensitivity and specificity minus 1. At the optimal cut-off, we estimated sensitivity, specificity, positive and negative predictive values, overall classification accuracy, positive and negative likelihood ratios, and the Youden index. Bootstrap resampling with 1,000 replications was used to obtain confidence intervals for the diagnostic accuracy measures.

Logistic regression was used as a companion analysis to estimate the association between linear growth velocity and stunting at 24 months. The unadjusted model included early linear growth velocity as the only predictor. A multivariable prediction model was then fitted to assess whether early growth velocity contributed to prediction after accounting for baseline and contextual risk factors. The adjusted model included early linear growth velocity, baseline stunting status, infant sex, martenal height, parity, HIV exposure status,the number of diarrhoeal adverse events before 24 months, and type of household toilet facility. Model discrimination was evaluated using ROC curves and AUC estimates. The incremental contribution of early linear growth velocity to the adjusted model was assessed by comparing nested models with and without early growth velocity using a likelihood-ratio test, fitted on the same complete-case estimation sample. Secondary logistic regression and ROC analyses were also conducted for each later interval-specific growth velocity measure to examine whether predictive performance varied across age windows.

To characterise longitudinal growth-velocity trajectories, the interval-specific velocity variables were reshaped into long format, with each child contributing one record per observed growth interval. Age was represented using the midpoint of each interval: 1.5, 4.5, 7.5, 10.5, 13.5, 16.5, 19.5, and 22.5 months. Observed mean growth velocities and corresponding 95% confidence intervals were plotted by interval and by stunting status at 24 months. Mixed-effects linear regression models were then fitted to estimate interval-specific growth velocity trajectories while accounting for repeated measurements within children. The primary trajectory model included fixed effects for 24-month stunting status, growth interval, and their interaction, with a random intercept for each child. The interaction between stunting status and growth interval was tested jointly to assess whether the shape of the growth velocity trajectory differed between children who were stunted and those who were not stunted at 24 months.

Sensitivity analyses were conducted to assess the influence of extreme or implausible interval-specific growth velocity values. Implausible velocities were flagged when interval growth velocity was less than –2 cm/month or greater than 8 cm/month. The mixed-effects trajectory models were repeated after excluding these implausible interval observations. Agreement between the primary and sensitivity analyses was assessed by comparing the estimated trajectories, interval-specific predicted means, and the joint test of the interaction between stunting status and growth interval.

Finally, restricted cubic spline mixed-effects models were fitted to explore nonlinear growth velocity patterns over time. Spline knots were placed at selected percentiles of the observed age-midpoint distribution. The spline models included 24-month stunting status, spline terms for age, and interactions between stunting status and the spline terms, with a random intercept for each child. A joint test of the spline-by-stunting interaction terms was used to assess whether the overall nonlinear growth velocity trajectory differed by 24-month stunting status. A sensitivity spline model was also fitted after excluding implausible interval velocities. All statistical tests were two-sided, and statistical significance was assessed at the 5% level. All statistical analyses were performed using Stata MP 19.5 (StataCorp, College Station, TX, USA).

3. Results

Background characteristics and participant flow

Between September 2018 and July 2021, 212 infants were enrolled into the ROVAS II study and followed up for over 24 months. Of which only 154 were between 6 and 8 weeks old, and had visit data at baseline, 3 months and 24 months. However, 34 infants had no outcome data at the timepoints of interest. Consequently, only 120 infants were used in the final analysis (Figure 1).

120 participants were included in the study, as shown in Figure 1. The median age at enrolment was 6 weeks, and 55% (n=66) were male. Only 4% (n=5) had a low birth weight, while 6% (n=7) were malnourished and 16% (n=19) were stunted. The mean middle-upper arm circumference (MUAC) was 12.4 cm (SD=0.9). About a third were HIV exposed but uninfected (HEU). Around half (52%, n=62) of the participants received a booster dose of Rotarix™ at 9 months. The mothers were on average 1.6 meters tall, with over half being of low parity (1-2 children). The majority of mothers had primary education or none (n=95, 79%), were not working (n=87, 73%), and were married or cohabiting (n=96, 80%). About 69% (n=83) earned less than ZMW1,000 a month, and 82% (n=97) of the households had unimproved toilets, with over 78% (n=94) sharing toilet facilities (Table 1).

Table 1: Participant characteristics

Characteristic	Total N = 120
	n (% of total)
Infant sex	
Female	54 (45.0)
Male	66 (55.0)
Infant age in weeks, Median (IQI[1])	6 (6-6)
Low birth weight (<2.5kg)	
No	115 (95.8)
Yes	5 (4.2)
Infant malnutrition status	
Not Malnourished	113 (94.2)
Malnourished	7 (5.8)
Infant stunting status	
Not Stunted	101 (84.2)
Stunted	19 (15.8)
Middle upper arm circumference (cm), Mean (Std. Dev[2])	12.4 (0.9)
HIV exposed	
HUU[3]	83 (69.2)
HEU[4]	37 (30.8)
Booster ROTARIX™ dose	
No	58 (48.3)
Yes	62 (51.7)
Mothers Characteristics	

Mother's height in meters, Mean (Std. Dev)	1.6 (0.1)
Parity	
Low Parity (1-2)	66 (55.0)
Moderate Parity (3-4)	36 (30.0)
High Parity (5+)	17 (14.2)
Missing	1 (0.8)
Mother's highest level of education	
None	26 (21.7)
Primary	69 (57.5)
Secondary/Tertiary	25 (20.8)
Mother's age category in years	
18-23	50 (41.7)
24-27	26 (21.7)
28-32	24 (20.0)
33+	20 (16.7)
Mother's employment status	
Student/Unemployed	87 (72.5)
Self-employed	27 (22.5)
Formally employed	6 (5.0)
Mother's marital status	
Single	24 (20.0)
Cohabiting/Married	96 (80.0)
Gestation	
Pre-term	5 (4.2)
Full term	115 (95.8)

Household Characteristics	
Monthly income	
< ZMW1,000	83 (69.2)
≥ ZMW1,000	36 (30.0)
Missing	1 (0.8)
Source of drinking water	
Unprotected/Protected well	7 (5.8)
Public tap/standpipe/borehole	77 (64.2)
Piped into the yard/house	36 (30.0)
Toilet facility	
Unimproved[5]	98 (81.7)
Improved[6]	22 (18.3)
Shared toilet	
No	26 (21.7)
Yes	94 (78.3)

1 = Interquartile interval, 2 = Standard Deviation, 3 = HUU HIV unexposed uninfected, 4 = HEU HIV exposed uninfected

5 = pit latrines without a slab or open pits, shared toilets and no toilet facilities

6 = flushing toilets, ventilated improved pit latrines and pit latrines with slab

Predictive accuracy of stunting at 24 months using early growth velocity

Early linear growth velocity showed only modest discrimination for identifying children who were stunted at 24 months. The ROC curve for early growth velocity lay only slightly above the line of no discrimination, with an area under the curve of 0.576. The corresponding unadjusted logistic prediction model based on early growth velocity alone gave a very similar AUC of 0.576, indicating that early linear growth velocity, on its own, had limited ability to distinguish children who subsequently became stunted from those who did not. In contrast, the adjusted early prediction model demonstrated substantially better discrimination. After incorporating early growth velocity together with baseline stunting and selected child, maternal, and household covariates, the AUC increased to 0.801. This suggests that early growth velocity contributes more meaningfully when interpreted alongside other baseline risk indicators rather than as a standalone screening marker (Figure 2 and Table 2). The optimal cut-off value for early linear growth velocity between enrolment and 3 months was approximately 2.99 cm/month, as determined using the Youden Index. Infants with a growth velocity at or below this threshold were classified as having low early linear growth velocity and were considered at increased risk of stunting at 24 months. This cut-off provided the best balance between sensitivity and specificity for predicting later stunting.

Table 2: Diagnostic accuracy of early linear growth velocity for predicting stunting at 24 months

Measure	Estimate	95% CI
Optimal cut-off (cm/month)	2.99	-
Sensitivity	0.517	0.389-0.645
Specificity	0.633	0.513-0.754
Positive predictive value (PPV)	0.577	0.441-0.713
Negative predictive value (NPV)	0.576	0.458-0.694
Accuracy	0.576	0.489-0.663
Positive likelihood ratio (LR+)	1.411	0.780-2.041
Negative likelihood ratio (LR−)	0.762	0.509-1.016
Youden Index	0.151	-

Secondary analyses of later interval-specific growth velocities showed that predictive performance varied by age window. Growth velocity from 3–6 months had weak discrimination for 24-month stunting, with an AUC of 0.528 and no evidence of association in logistic regression (OR = 0.85; 95% CI: 0.51–1.41; p = 0.525). Growth velocity from 6–9 months performed no better than chance, with an AUC of 0.501 and no association with later stunting (OR = 1.05; 95% CI: 0.72–1.52; p = 0.812). In contrast, growth velocity from 9–12 months showed better discrimination, with an AUC of 0.659, and was inversely associated with stunting at 24 months (OR = 0.45; 95% CI: 0.26–0.77; p = 0.003). This indicates that higher growth velocity during 9–12 months was associated with substantially lower odds of being stunted at 24 months.

The later growth windows showed mixed predictive patterns. Growth velocity from 12–15 months had poor discrimination, with an AUC below 0.5 (AUC = 0.425), and was not significantly associated with 24-month stunting (OR = 1.35; 95% CI: 0.77–2.36; p = 0.289). Growth velocity from 15–18 months showed weak-to-moderate discrimination (AUC = 0.564), but the association did not reach conventional statistical significance (OR = 0.68; 95% CI: 0.41–1.11; p = 0.119). Growth velocity from 18–21 months had no discriminatory value (AUC = 0.504) and no association with stunting (OR = 1.00; 95% CI: 0.62–1.61; p = 0.995). The strongest interval-specific discrimination was observed for growth velocity from 21–24 months, which had an AUC of 0.700 and was strongly inversely associated with stunting at 24 months (OR = 0.29; 95% CI: 0.14–0.63; p = 0.002). This finding is expected because this interval is closest in time to the outcome assessment and therefore reflects more proximal growth faltering.

Longitudinal growth velocity patterns

The observed growth velocity trajectories showed a steep decline in linear growth velocity across infancy, with the highest velocities occurring in the earliest interval and substantially lower velocities thereafter. Children who were not stunted at 24 months generally had higher observed mean growth velocity than stunted children, particularly during the earliest interval and again toward the end of follow-up. However, the two trajectories were not widely separated across all intervals, suggesting that growth faltering was not confined to a single discrete age period but reflected a broader pattern of lower linear growth across infancy and early childhood (Figure 3 Panel A).

The model-based interval-specific predicted trajectories were consistent with the observed pattern. Predicted growth velocity was highest in the early infancy interval, declined sharply by 6–9 months, and then remained relatively low through 24 months. Across most intervals, children who were stunted at 24 months had lower predicted growth velocity than those who were not stunted. The clearest separations were seen during early infancy, around 9–12 months, and toward 21–24 months. The overall interaction between stunting status and interval was borderline in the primary mixed-effects model ($\chi^2 = 13.35$; $p = 0.064$), suggesting some evidence that the shape of the growth velocity trajectory differed by 24-month stunting status, although this did not meet the conventional 5% significance threshold (Figure 3 Panel B).

In the primary mixed-effects model, children who were stunted at 24 months had, on average, lower growth velocity at the reference interval than children who were not stunted (coefficient = −0.34 cm/month; 95% CI: −0.66 to −0.03; $p = 0.031$). Predicted growth velocity in the 0–3-month interval was 3.46 cm/month among children not stunted at 24 months and 3.12 cm/month among those stunted at 24 months. Both groups experienced a marked decline after early infancy. By 9–12 months, predicted velocity was 1.09 cm/month among children not stunted and 0.65 cm/month among children stunted at 24 months. At 21–24 months, predicted velocity was 0.91 cm/month among children not stunted compared with 0.50 cm/month among children stunted at 24 months. These estimates indicate that children who were stunted at 24 months tended to have persistently lower linear growth velocity, particularly during selected later intervals (Figure 3 Panel C).

The sensitivity analysis excluding implausible interval velocities produced results that were highly consistent with the primary trajectory model. The estimated mean difference at the reference interval remained similar (coefficient = −0.34 cm/month; 95% CI: −0.65 to −0.04; $p = 0.028$), and the interval-by-stunting interaction remained borderline ($\chi^2 = 12.56$; $p = 0.084$). The predicted trajectories in the sensitivity graph closely overlapped those from the primary analysis, indicating that the overall findings were not driven by a small number of extreme or implausible growth velocity observations (Figure 3 Panel C).

The spline-smoothed trajectory graphs further supported a nonlinear pattern of postnatal growth velocity. In both the primary and sensitivity spline models, growth velocity declined rapidly from early infancy to around 9–12 months, followed by a modest flattening and slight recovery during the second year of life before declining again toward 24 months. The smoothed curves for children who were stunted at 24 months remained generally below those for children who were not stunted. However, the formal spline interaction terms did not show evidence that the overall smoothed trajectory shape differed significantly by stunting status in either the primary model ($\chi^2 = 1.79$; $p = 0.616$) or the sensitivity model excluding implausible velocities ($\chi^2 = 1.74$; $p = 0.629$). This suggests that although children who became stunted tended to have lower growth velocity, the broad nonlinear pattern of growth velocity over time was similar in both groups (Figure 3, Panels D and E).

Stunting dynamics during the first 24 months of life

At 6-8 weeks, approximately 85% of the participants had never experienced stunting. At 3 months, approximately 10% were stunted, steadily increasing, with the largest increase seen between 9 and 15 months. A small proportion of stunted infants showed signs of partial recovery before the age of 24 months, while a smaller proportion also showed signs of relapse, showing that once stunted, some participants remain persistently stunted through to 24 months. By 24 months, only about 30% children had not experienced any stunting. This shows that stunting is a dynamic process rather than a state at a particular time point (Figure 4).

4. Discussion

Summary of key findings

Early post-enrolment growth velocity showed only modest discrimination for subsequent stunting, with the ROC curve lying only slightly above the line of no discrimination and the unadjusted logistic model indicating limited predictive value. In contrast, the adjusted early prediction model demonstrated substantially better discrimination, suggesting that early growth velocity alone is not sufficient as a standalone screening marker but may add value when considered alongside baseline stunting status and other child and maternal characteristics. Analyses of later growth intervals showed stronger evidence for associations during 9–12 months and 21–24 months, where lower velocity was associated with higher odds of being stunted at 24 months. Longitudinal trajectory analyses showed a marked decline in growth velocity after early infancy, followed by lower and relatively stable velocities through the second year of life. Children who were stunted at 24 months generally had lower predicted growth velocity than those who were not stunted, particularly in early infancy and toward the end of the second year, although formal tests provided limited evidence of a big overall difference in velocity trajectories by later stunting status. These findings suggest that stunting at 24 months is not adequately explained by a single early velocity measure, but rather reflects a broader pattern of growth performance across infancy and the second year of life.

Interpretation of findings

The modest discrimination of early linear growth velocity alone suggests that a single short-interval velocity measure, taken soon after enrolment, may not adequately capture the cumulative biological and social processes that lead to stunting by 24 months. This is consistent with evidence that linear growth faltering often begins very early, including during the prenatal period and the first months of life, and that stunting onset and persistence are shaped by repeated exposures rather than by one isolated postnatal growth interval. In a pooled analysis of 32 longitudinal cohorts from low- and middle-income countries, Benjamin-Chung and colleagues found that the highest incidence of stunting onset occurred from birth to 3 months, while reversal was uncommon from 0 to 15 months and relapse was frequent among children who reversed stunting status. They also reported that, after 3 months of age, average length velocity in many cohorts was approximately 0.75–1.25 cm/month, indicating that later growth increments are small and may be less able to compensate for earlier deficits (Benjamin-Chung et al., 2023). Our finding that the adjusted early prediction model performed substantially better than early velocity alone is therefore biologically plausible. Baseline nutritional status, maternal stature, child characteristics, and household-level conditions are likely to contain information on prenatal growth, early-life adversity, and underlying vulnerability that is not fully captured by early velocity. This interpretation is also supported by evidence from Zambia. In a prospective cohort of infants followed from 6 weeks to 24 months at Kamwala Urban Health Centre in Lusaka, Chilengi and colleagues found that infant linear growth velocity was consistently below the WHO growth velocity standard, with growth faltering beginning early and becoming most pronounced around 13.6 months (Chilengi et al., 2019). Thus, our results align with evidence showing that growth faltering is already established during infancy and may reflect a broader population-level growth deficit rather than a process that can be reliably identified using one early velocity measure alone.

The stronger associations observed for the 9–12 month and 21–24 month velocity intervals indicate that growth during the latter part of infancy and the second year of life remains important for attained HAZ at 24 months. The inverse associations in these intervals, where higher velocity was associated with lower odds of stunting, are consistent with the expectation that children who maintain or recover linear growth during complementary feeding and late infancy are less likely to be stunted at 24 months. However, the weak or null associations in several other intervals suggest that interval-specific velocity is variable and may be influenced by measurement error, short-term illness, feeding transitions, and regression to the mean. The trajectory graphs support this interpretation. Growth

velocity declined sharply after early infancy and remained low thereafter, with children who became stunted generally showing lower predicted velocity, although the overall trajectory differences were not strongly separated. This pattern agrees with global analyses showing that the most pronounced faltering occurs within the first 1000 days, particularly between 6 and 18 months, but that growth deficits may continue accumulating beyond age 2 years when assessed in absolute height terms (Leroy et al., 2014). Evidence also points to the multidimensional nature of stunting. Mzumara and colleagues, using nationally representative Zambian data, identified child age and sex, maternal age and education, household wealth, water source, breastfeeding duration, and residence as major predictors of stunting among children under five (Mzumara et al., 2018). Our findings suggest that linear growth velocity is informative when interpreted as part of a broader growth and risk profile, but it should not be treated as a standalone screening tool for future stunting. Programmatically, the results reinforce the need for early identification of children with poor attained growth, continued monitoring through the complementary feeding period, and interventions that address maternal nutrition, infant feeding, infection prevention, and household socioeconomic conditions.

Strengths and Limitations

This study has several strengths. First, it used longitudinal child-level anthropometric data with repeated measurements across infancy and early childhood, allowing growth velocity to be examined across multiple age intervals rather than relying only on attained HAZ at a single time point. Second, stunting was defined directly from HAZ using the conventional WHO threshold of HAZ < −2, and growth velocity was calculated from measured length/height and age, thereby ensuring that the main exposure and outcome were derived transparently from primary anthropometric information. Third, the analysis treated early linear growth velocity as both a screening and predictive marker, using ROC analysis, Youden-derived cut-off estimation, diagnostic accuracy measures, and companion logistic regression models; this provided a more complete assessment of clinical and public health usefulness than an association-only analysis. Fourth, the study evaluated both unadjusted and adjusted prediction models, showing how the addition of baseline stunting, child sex, parity, adverse events, and maternal height improved discrimination. Finally, the longitudinal trajectory analysis, including sensitivity analyses excluding implausible interval velocities, provided a useful description of how growth velocity varied over time among children who were and were not stunted at 24 months.

The study also has limitations. The sample size was modest, which reduced precision around estimates of sensitivity, specificity, likelihood ratios, and interval-specific associations. Some children had missing measurements at particular time points, resulting in variable sample sizes across growth-velocity intervals and potentially limiting comparability between intervals. Because visits did not occur exactly at the target ages, interval velocities depended on the closest available measurements within prespecified age windows. This may have introduced measurement variability, particularly where the time difference between observations was short. Length and height measurements in young children are also prone to random error, which may explain some negative or extreme interval velocities and may have attenuated associations. In addition, although the adjusted model improved prediction, it was developed and assessed in the same dataset, so its apparent performance may be optimistic and requires external validation before use as a screening or risk-stratification tool.

Implications for future research and practice

The findings have important implications for both future research and practice. For practice, early linear growth velocity alone should not be used as a stand-alone screening tool for identifying children who will become stunted by 24 months, given its modest discrimination and limited diagnostic accuracy. However, routinely tracking growth

velocity during infancy may still add value when interpreted alongside attained HAZ, baseline nutritional status, child morbidity, maternal height, and other household or clinical risk factors. In child health and nutrition programmes, this supports a shift from relying only on cross-sectional growth status to using repeated anthropometric measurements to identify children whose growth pattern is faltering before stunting becomes established. The stronger performance of the adjusted prediction model suggests that practical risk stratification tools should combine growth velocity with simple clinical and maternal indicators that are already collected in routine maternal and child health services. For research, larger longitudinal studies are needed to validate these findings, refine age-specific growth-velocity thresholds, and assess whether prediction models developed in this setting perform well in other Zambian and sub-Saharan African populations. Future studies should also examine whether incorporating dietary intake, infection history, birthweight, maternal nutrition, household food security, water and sanitation indicators, and socioeconomic conditions improves prediction. Most importantly, future implementation research should evaluate whether children identified as high risk through combined growth monitoring and risk prediction can benefit from earlier, targeted nutrition and health interventions before linear growth failure becomes irreversible.

5. Conclusion

Early linear growth velocity was associated with stunting at 24 months, but its value as a stand-alone screening marker was modest. The optimal early velocity cut-off had limited sensitivity and specificity, and the unadjusted model showed weak discrimination. Prediction improved when early growth velocity was combined with baseline stunting, child sex, parity, diarrhoeal episodes, HIV status, maternal height, and type of toilet facility, suggesting that stunting risk is better captured through a multivariable approach. Longitudinal analyses showed that children stunted at 24 months generally had lower growth velocity across infancy and early childhood, with clearer separation during selected later intervals. These findings support repeated growth monitoring while cautioning against reliance on early growth velocity alone. In Zambia and similar settings, integrating growth velocity with routinely collected maternal, child, and morbidity indicators may strengthen early risk identification and guide timely nutrition interventions.

Declarations

Author contributions

Conceptualisation B.P., M.S, M.C; methodology, B.P, P.M, S.B; analysis, B.P; data acquisition, B.P; data interpretation and manuscript drafting and review, B.P, M.S, N.L, M.C, S.B, C.M, P.M, R.C, M.C; data visualisation, B.P. All authors have read and agreed to the published version of the manuscript.

Funding

This research was funded in part by the Science for Africa Foundation through the Developing Excellence in Leadership, Training and Science in Africa (DELTAS Africa) programme, grant number DEL-22-012. This study was also partly funded by the ELMA Cholera Relief Foundation.

Institutional Review Board Statement

Ethical approval was obtained from the University of Zambia Biomedical Research Ethics Committee (UNZABREC) (reference number 001-02-23) and the National Health Research Authority (NHRA).

Consent to Participate

Written informed consent was obtained from the parents or legal guardians of all participating infants before enrolment in the parent study.

Clinical Trial Registration

The parent ROVAS II trial was registered with the Pan African Clinical Trial Registry (PACTR) (Ref: PACTR201804003096919 | https://pactr.samrc.ac.za/TrialDisplay.aspx?TrialID=3096) registered on February 16, 2018.

Consent for Publication

Not applicable.

Data Availability Statement

Data are available to qualified researchers upon request, subject to approval by the CIDRZ Ethics and Compliance Committee. Requests should be emailed to Ms. Hope Chinganya, Secretary to the Committee and Head of Research Operations (hope.chinganya@cidrz.org), and must specify the requester's contact details, project title, planned analyses, and desired data format. Data use is restricted to purposes aligned with the original study; the Committee typically reviews submissions within 48–72 hours (business days), notifying applicants of approval status or any required additional details.

Acknowledgments

The authors gratefully acknowledge the infants and their families who participated in this study, as well as the study team and field staff whose dedication made data collection and follow-up possible. Sincere appreciation is extended to supervisors, colleagues, and reviewers for their valuable scientific guidance and constructive feedback during the development of this manuscript. The authors also acknowledge the institutions and organisations that supported the original study and the academic support provided to B.P through the MSc in Medical Statistics programme.

Competing Interests

The authors declare that they have no competing interests.

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Figures

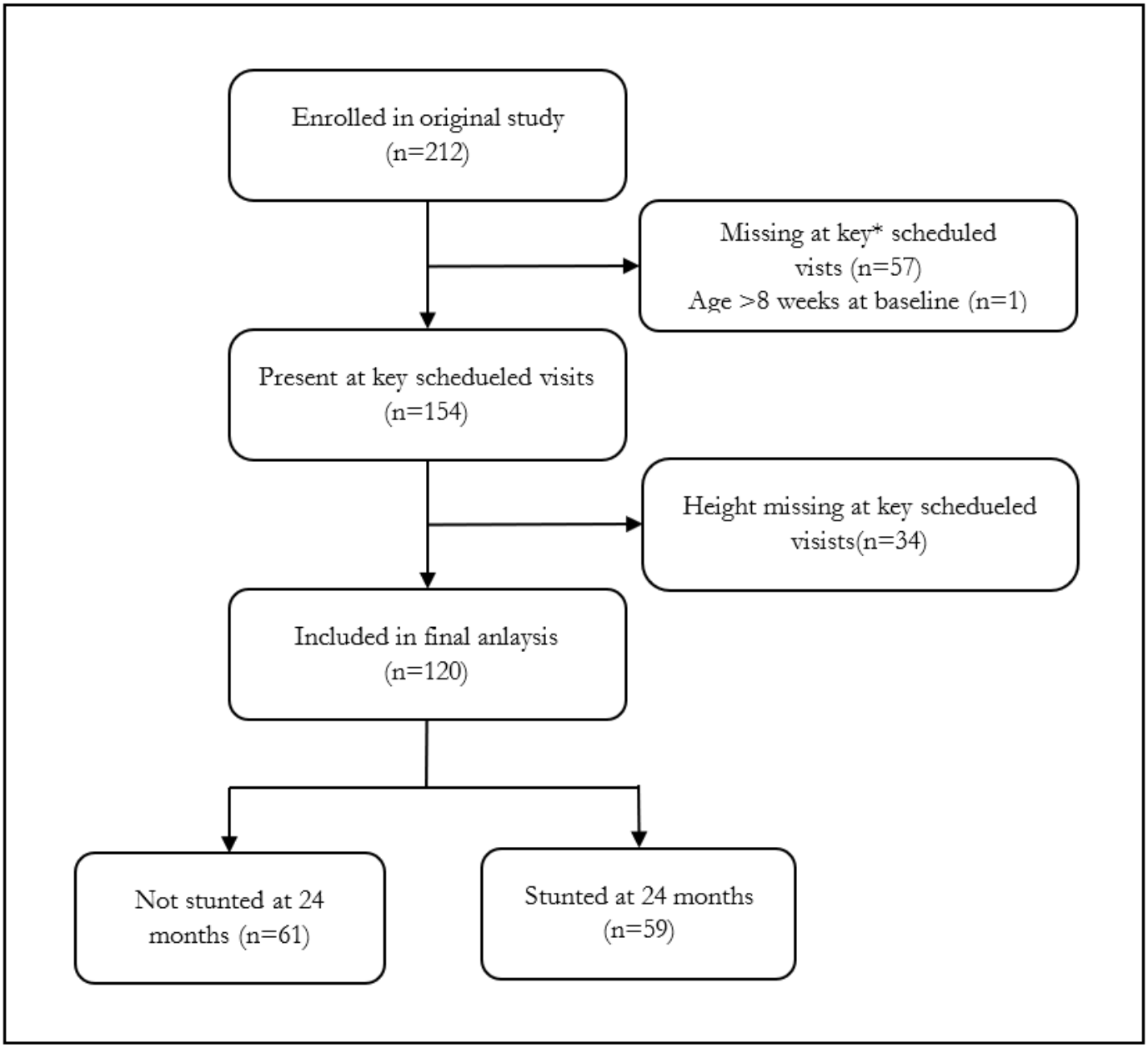


Figure 1

Participant flow, ** key scheduled visits include baseline, 3 months and 24 months*

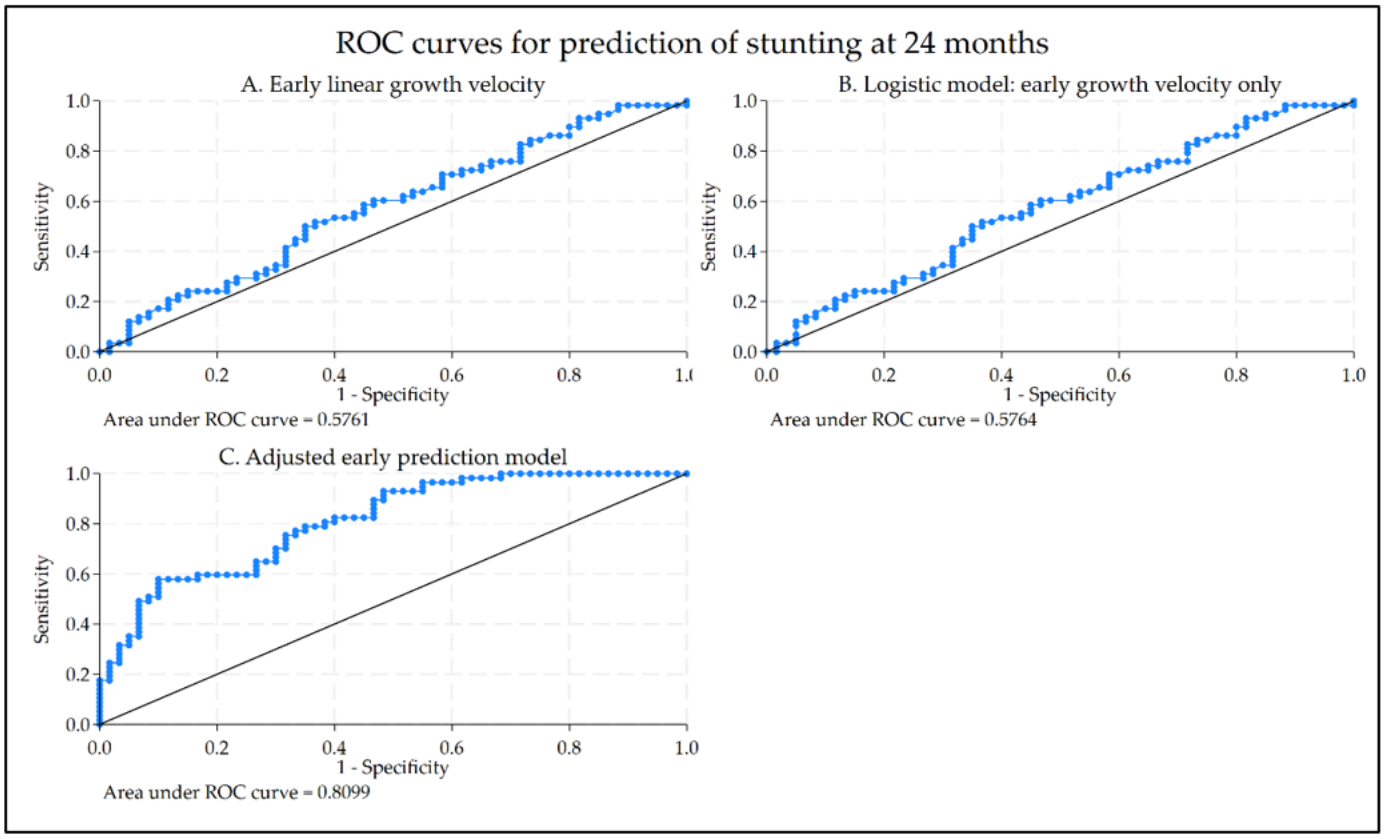


Figure 2

ROC curves for the prediction of stunting at 24 months. Panel A shows the ROC curve for early linear growth velocity between 0 and 3 months. Panel B shows the ROC curve for the unadjusted logistic regression model, including early linear growth velocity only. Panel C shows the ROC curve for the adjusted prediction model incorporating early linear growth velocity, baseline stunting status, infant sex, parity, maternal height, HIV exposure status, type of toilet facility, and number of diarrhoeal adverse events before 24 months.

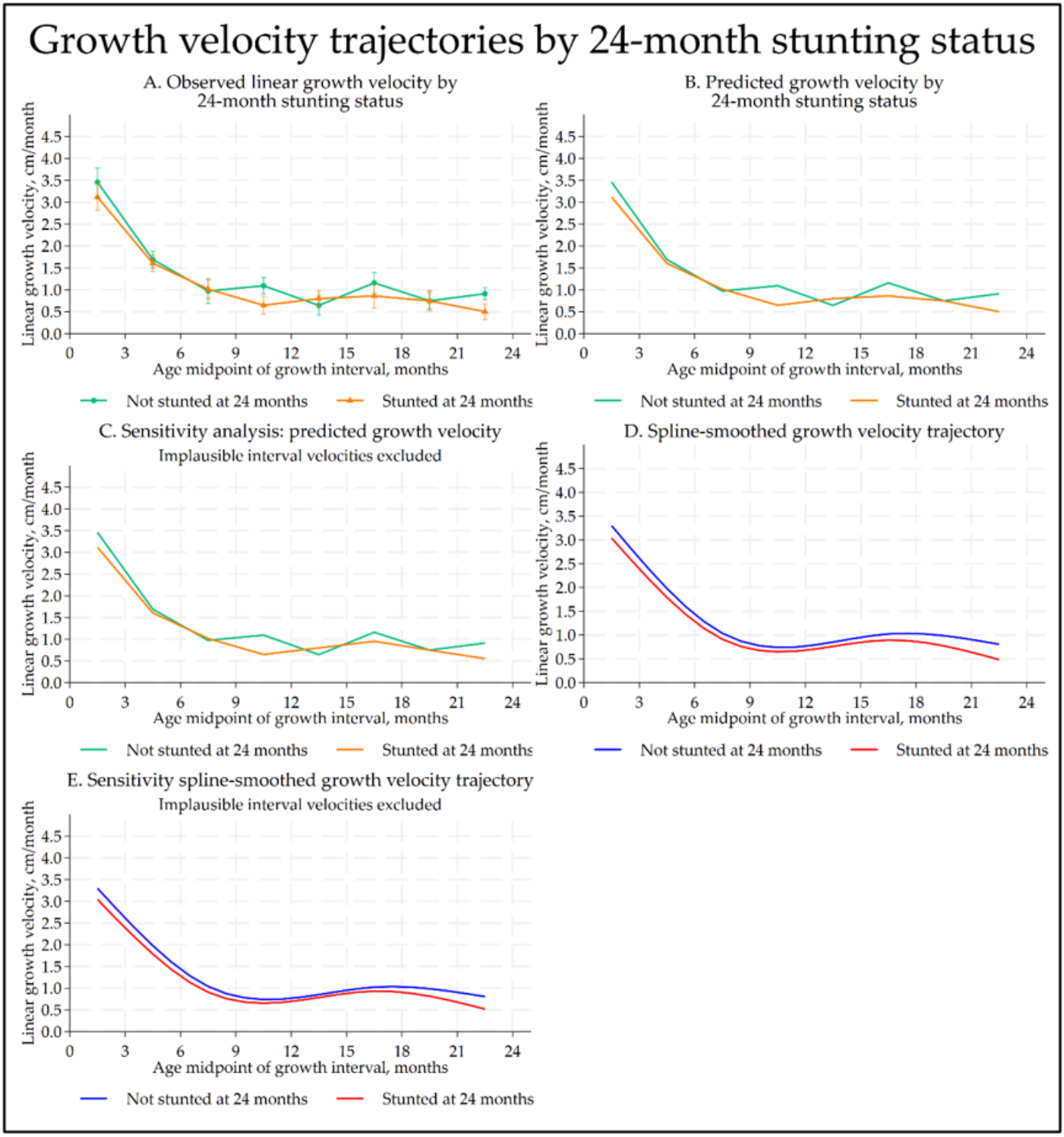


Figure 3

Longitudinal linear growth velocity trajectories according to stunting status at 24 months. Panel A shows observed mean linear growth velocity across age intervals stratified by 24-month stunting status. Panel B presents predicted interval-specific growth velocity trajectories from the primary mixed-effects model. Panel C shows predicted growth velocity trajectories from the sensitivity mixed-effects model excluding implausible interval velocities. Panel D presents spline-smoothed growth velocity trajectories from the primary spline mixed-effects model. Panel E shows spline-smoothed trajectories from the sensitivity spline model excluding implausible interval velocities. Across most

intervals, children who were stunted at 24 months demonstrated lower growth velocity compared with children who were not stunted.

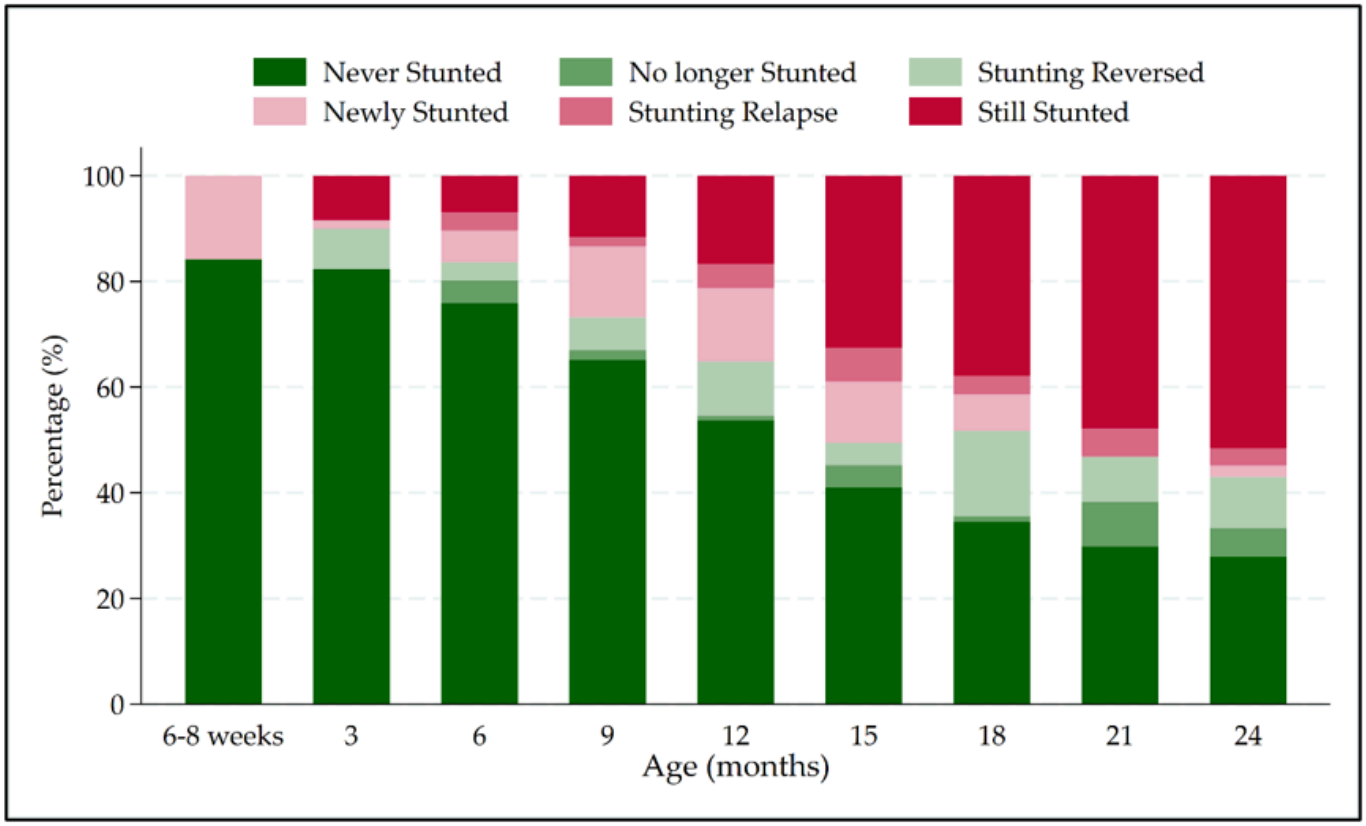


Figure 4

Percentage of children with stunting reversal and relapse by age. The categories are defined as: Never stunted - never experienced stunting at all previous ages and current age; No longer stunted - previously reversed their stunting status and remained non-stunted (HAZ ≥−2) at the current age; Stunting reversal - stunted at previous age but had HAZ ≥−2 at the current age; Newly stunted (first stunting) - never stunted at any previous ages and HAZ <−2 at the current age; Stunting relapse - previously stunted, had HAZ ≥−2 at the previous age and HAZ <−2 at the current age; Still stunted - HAZ <−2 at the previous and current age.